as an arresting and cardioprotective agent, and the concomitant use of a local anesthetic.

A major object of our study was to investigate whether this combination was efficient in normokalemic cardioplegia, because possible negative effects of hyperkalemia have been debated for decades and normokalemic adenosine/local anesthetic cardioplegia has been shown to be superior in animal studies, compared with both high and moderate potassium concentrations.¹ Despite some clinical studies with the use of adenosine in the cardioplegic solution, to our knowledge, this is the first study on the use of normokalemia.

As for the choice of local anesthetic, we used procaine because our standard hyperkalemic cardioplegia derived from St Thomas' Hospital solution contains 1 mmol/L of procaine to reduce the group differences. Both procaine and lidocaine reduce the action potential amplitude and the maximum rate of increase of the upstroke of the action potential, but they differ in the influence of the action potential duration (APD), where procaine increases and lidocaine shortens the APD. They both have rhythm-stabilizing effects after cardiac surgery. Cardioprotective properties, although dose-dependent, have been demonstrated for both drugs in isolated heart studies.² Although larger clinical studies exclusively comparing these 2 local anesthetics in cardioplegia are missing, we agree that adenosine-procaine and adenosine-lidocaine cardioplegia are probably 2 sides of the same coin.

What is most important in achieving cardioplegic arrest: adenosine, magnesium, or Na⁺ channel blockers, such as lidocaine or procaine? Hypothermia is the largest contributor to the arrest, and infusing 750 mL of cold crystalloid solution is going to arrest the heart almost regardless of composition. Whether this solution will protect the heart while in its

resting state or not is another matter. It is not a given that cardiac arrest and cardiac protection are equivalent. Biochemical processes are highly temperature-dependent, and little research has been done to assess the behavior of drugs in hypothermic conditions like the heart is in cardiac surgery. We have previously shown that hypothermia actually reversed the APD shortening of adenosine triphosphate-sensitive K⁺ (K_{ATP}) channel openers, thus theoretically making the K_{ATP} channel openers less effective in achieving cardiac arrest under hypothermic conditions. Nevertheless, the K_{ATP} openers provided cardioprotective properties even in hypothermic conditions.³ We also previously showed that adenosine has no effect on ventricular contractility at normothermia, whereas 1.2 mmol/L of adenosine significantly reduces contractile force at moderate (24°C) hypothermia.⁴

There are now several promising studies from the use of adenosine in cardioplegia, so alternatives to potassium arrest may be in the making. However, several issues still should be addressed regarding the composition, optimal doses, type of delivery, and vehicle. For instance, what are the optimal dose, temperature, and administration of adenosine in crystalloid and blood cardioplegia? What is the role of adenosine in preconditioning? Steensrud and colleagues⁵ have shown that adenosine can induce remote preconditioning when given intra-arterially, but not intravenously, and that this effect is dependent on an intact nerve system but inhibited by nitric oxide. Thus, concomitant medications, such as nitric oxide donors or sulfonyleureas and even anesthesia, may play a role in the search of improving the current cardioprotective solution. In the future, we may be able to individualize our cardioplegic strategy according to the patient's needs, that is, if alternatives exist.

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FOLDING OR PLICATION TECHNIQUE IN MITRAL VALVE REPAIR: NEW OR RENAMED?

To the Editor:

It was with great interest that we read the article by Tsukui and colleagues¹ on early outcome of the folding mitral valve repair technique without resection for mitral valve prolapse in 60 patients. This folding mitral valve repair technique—or FMVR, as the authors call it—is described as a non-resectional technique with inversion of the prolapsed segment into the left ventricle and ring annuloplasty, also applied for prolapse secondary to ruptured chordae. The authors apply a pilot stitch vertically to the edge of the prolapsed segment of the posterior leaflet, and additional

interrupted sutures are placed toward the annulus.

We agree with Dr Miller's comment in the discussion that this is not a new technique. It is true that it is highly similar to McGoon's technique of triangular plication of the prolapsing portion of the posterior leaflet resulting from ruptured chordae, which was published in 1960,² not in 1985, as suggested by the reference misquoted by Tsukui and colleagues.¹ (Reference 12 by Tsukui and colleagues to "McGoon DC. Repair of mitral insufficiency due to ruptured chordae tendineae. *J Thorac Cardiovasc Surg.* 1985;89:491-8" seems to be an incorrect rendering of Orszulak TA, Schaff HV, Danielson GK, Piehler JM, Pluth JR, Frye RL, et al. Mitral regurgitation due to ruptured chordae tendineae: early and late results of valve repair. *J Thorac Cardiovasc Surg.* 1985;89:491-8.)

McGoon, of course, did not perform ring annuloplasty because rings were not introduced until 9 years after he published his technique.

Gerbode and colleagues³ performed plication of the posterior leaflet for posterior leaflet prolapse resulting from ruptured chordae tendinae and first published it in 1962. Plication involves 4 layers of leaflet tissue, 2 on each side, which adds to stability of the plication line, in contrast to suture after resection, which involves 2 layers and is more prone to rupture. Through the years, this technique has been modified. Plication of the posterior leaflet toward the ventricle with interrupted mattress sutures and addition of posterior annuloplasty with stabilization of the repair by an autologous pericardial strip has become the standard concept for mitral valve repair at our center for posterior leaflet prolapse, for ruptured chordae tendinae, and even in repair for endocarditis.^{4,5} Tsukui and colleagues¹ unfortunately seem to be unaware of these references.

With regard to Miller's comment on why surgeons are trying to reincarnate

old techniques, this plication technique has, in fact, not been abandoned nor forgotten, but continues to show good long-term functional outcome. The folding mitral valve repair technique used by Tsukui and colleagues¹ is, therefore, not new, but another version of the modification by Gerbode and colleagues³ of the original McGoon² plication principle. Further modifications have been continuously proposed and tested, including our contributions.^{4,5}

It seems to us that the issue here is purely semantic and not technical. Hence, to allege that the technique described by Tsukui et al¹ is innovative is misleading.

We thank Anne Gale, ELS, for editorial assistance.

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Reply to the Editor:

I thank Drs Hetzer and Walter for their letter regarding our article in the January 2013 issue of the *Journal*. I am embarrassed to note that our article failed to reference McGoon's article, published in 1960, describing the

technique for triangular plication of the prolapsing posterior leaflet caused by ruptured chordae tendineae. Also, I thank Hetzer and Walter for reminding us of previous works from Gerbode and colleagues and from their own institution. I have enjoyed these articles.

Our mitral valve repair technique, folding mitral valve repair, does seem to be similar to other techniques reported by several authors; however, what we intended to emphasize in our article is the simplicity and reversibility of this technique, rather than its novelty. We expect these features to contribute to a greater repair rate and to provide a chance for residents to gain experience in mitral valve repair. The novel aspect of our technique is the expansion of indication to the anterior leaflet, not A2, and the commissural leaflet after successful experiences with the posterior leaflet. To the extent of my research, there have been no articles reporting detailed outcomes of anterior and commissural leaflet repair with plication or folding techniques, such as we reported.

Finally, I appreciate the commentary of Drs Hetzer and Walter, and I look forward to reporting late outcomes of our technique in the future.

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IS GENDER-SPECIFIC SURVIVAL IN PATIENTS UNDERGOING RADIAL ARTERY GRAFTING INFLUENCED BY HORMONE LEVELS?

To the Editor:

We read with great interest the article by Dimitrova and colleagues¹ on